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Health and Environmental Effects Profile for 2-Propenoic Acid

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16. ABSTRACT

The Health and Environmental Effects Profile for 2-Propenoic Acid was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for 2-Propenoic Acid.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR 2-PROPENOIC ACID

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
NOEL	No observed effect level
ppm	Parts per million
TLV	Threshold limit value
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Acrylic acid is the common name for 2-propenoic acid. This chemical is also known as acroleic acid, ethylenecarboxylic acid, propenic acid and vinylformic acid (NIOSH, 1983; IARC, 1979). The structure of acrylic acid is given below:

$$CH_2=CHCOOH$$

Molecular formula: $C_3H_4O_2$

Molelular weight: 72.06

The Chemical Abstract Service (CAS) Registry number for acrylic acid is 79-10-7.

1.2. CHEMICAL AND PHYSICAL PROPERTIES

Acrylic acid is a colorless, corrosive, fuming liquid with an acrid odor and the following characteristics (Windholz, 1983; Verschueren, 1983):

Boiling point:	141°C
Melting point:	12-14°C
Vapor pressure:	3.2 mm Hg at 20°C
Specific gravity:	1.06 at 16°C
Vapor density:	2.5 (air = 1)
Solubility:	miscible with water, ether and ethanol; moderately soluble in acetone and benzene.
pK	4.3
Log octanol/water partition coefficient (K_{ow}):	0.31-0.43 (calculated)

1.3. PRODUCTION DATA

Two methods of preparing acrylic acid have been used in the United States: oxidation of propylene to acrolein with further oxidation to acrylic acid; or modifications of Reppe processes, which involve the use of

acetylene, alcohol and carbon monoxide gas (or nickel carbonyl), or acetylene, carbon monoxide and water with nickel halide as a catalyst (IARC, 1979).

The major producers of acrylic acid and their production capacities are summarized in Table 1-1. These producers convert most of the acrylic acid into ester monomers and functional acrylates (SRI, 1983).

1.4. USE DATA

Acrylic acid is used as a precursor for acrylates (used for surface coatings, textile application, and production of paper, polishes, acrylic fibers and leather), for the production of water-soluble resins and salts, and as a comonomer in acrylic emulsions and solution polymers (IARC, 1979).

TABLE 1-1

Major Producers of Acrylic Acid*

Producer	Location	Annual Capacity (millions of pounds)
Badische Corp.	Freeport, TX	130
Celanese Corp. Celanese Chem. Co., Inc.	Clear Lake, TX	300
Rohm and Haas Co., Rohm and Haas Texas, Inc.	Deer Park, TX	380
Union Carbide Corp. Coating Materials Division	Taft, LA	140

*Source: SRI, 1983

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Information regarding the fate and transport of acrylic acid in the environment is limited. Acrylic acid may enter the environment during manufacturing processes, and during handling or transportation.

The fate of acrylic acid in the atmosphere depends upon its ability to undergo chemical and photochemical reactions. According to U.S. EPA (1981), the acrylic acid monomer rapidly polymerizes in the presence of oxygen. In an abstract of a study by Kirchner et al. (1976), it was stated that the kinetics of the reaction of acrylic acid with ozone indicate rapid decomposition (no rate constants or half-lives were given).

Miyata and Arai (1981) found that the products of the reaction of acrylic acid with ozone were glyoxylic acid and formic acid with subsequent formation of hydrogen peroxide and citric acid. The miscibility of acrylic acid with water (Windholz, 1983) predicts that the compound could be removed from the atmosphere by rain.

The fate of acrylic acid in water will depend upon microbial degradation and chemical and photochemical reactions. Arai et al. (1981) reported that Rhodococcus sp. isolated from the effluent of an acrylamide plant converted acrylamide to acrylic acid, which in turn was converted to NH_3 and CO_2. Yamada et al. (1979) reported that a bacterium of the genus Arthobacter, isolated from acclimated activated sludge, could use acrylic acid as a sole carbon source. In an abstract of a Russian study, it was reported that in water artificially contaminated with acrylic acid, the biological oxygen demand (BOD) was quite high (Ekhina and Ampleeva, 1977). Sasaki (1978) indicated that acrylic acid is highly biodegradable since its BOD exceeded 30% after 2 weeks in active sludge. Dore et al. (1975) reported a chemical

oxygen demand (COD) of 100% for acrylic acid. These studies, while indicating that acrylic acid can be degraded microbially under controlled conditions, are difficult to relate to actual environmental conditions.

The polymerization of the acrylic acid monomer in the presence of oxygen may also occur in water, but at a slower rate than in air (U.S. EPA, 1981). The photoreactivity of acrylic acid is indicated in an abstract of a study by Pant et al. (1979). UV irradiation of acrylic acid in aqueous solutions with NH_3 or N_2 for 40 hours resulted in the formation of amino acids, a process which was inhibited by H_2.

The transport of acrylic acid in aquatic media will depend on the ability of the chemical to volatilize, to sorb to particulate matter or to be transported in the dissolved state in water. From the relatively low vapor pressure and low K_{ow} value (Verschueren, 1983) and high solubility (Windholz, 1983) of acrylic acid, one can predict that the chemical will be transported in the dissolved state in water rather than by volatilization or sorption to suspended solid matter in aquatic media.

The fate of acrylic acid in soil will also depend upon chemical, photochemical and microbial degradation processes. Although no studies were found in the literature cited in the Appendix, parallels may be drawn to the fate of acrylic acid in water. Although microbial degradation is possible (Yamada et al., 1979; Arai et al., 1981; Ekhina and Ampleeva, 1977; Sasaki, 1978; Dore et al., 1975), the controlled cirumstances of the studies make it difficult to relate the results to environmental conditions. In an abstract of a Japanese study (Nishikawa et al., 1979), it was reported that the acrylic acid formed from hydrolysis of acrylamide added to soils was itself totally degraded within 15 days of its formation. Photoreduction of acrylic acid may also occur at the soil surface.

Transport in soil will be governed by the high water solubility and low vapor pressure of acrylic acid. The chemical is not likely to sorb strongly to soil or to volatilize from it, but leaching to groundwater and runoff to surface water are more likely transport processes for this chemical in soil.

3. MONITORING-EXPOSURE

Pertinent monitoring data regarding acrylic acid were not located in STORET or in the available literature.

Human exposure to acrylic acid will occur primarily in the workplace. Unreacted acrylic acid monomer may be present in such products as polishes, paints, rug backing, adhesives, plastics, textiles and paper finishes (SRI, 1976). Serth et al. (1978) reported that in the United States in 1978, the total emissions of hydrocarbons into the atmosphere from acrylic acid production plants were estimated at <0.0006% of total national emissions from stationary sources. Emissions were not expected to increase in the future because of the high efficiency (98%) of plant incinerators. Estimates for acrylic acid, per se, were not available.

In 1977, 36.3 metric tons of acrylic acid emissions were reported from the production of n-butyl acrylate (118 thousand metric tons) (Eimitis and Quill, 1977). Acrylic acid emissions are likely to result from production of other acrylates, as well.

4. PHARMACOKINETICS

Kutzman et al. (1982) administered [^{11}C] acrylic acid, labeled on the carbonyl carbon, by nose-only exposure for 1 minute or by gavage as an aqueous solution to female Sprague-Dawley rats for a maximum dose of 26 µg/kg. The data presented were corrected for decay of the radioisotope (half-life = 20.4 minutes). The use of ^{11}C rather than the more conventional ^{14}C was apparently based on availability at the authors' laboratory.

Of the acrylic acid delivered to the nose cone, 18.3% was accumulated by the rats. Of this amount, 28.4% was associated with the exposed snout and 42.9% with the head region minus the exposed snout in rats sacrificed 1.5 minutes postexposure, indicating that inhaled acrylic acid was apparently solubilized in the mucus of the nasal turbinates and the naxophasynx of the exposed animals. Analysis of various tissues indicated that the highest initial levels of radioactivity occurred, in order of descending relative retention, in the lungs, kidney and intestinal contents. At 1 hour post-exposure, the relative retention of radio-activity was highest in the urine followed by stomach, intestinal contents, liver and kidney. The authors suggested that the increase at 1 hour of label associated with the stomach resulted from ingestion of the compound during postexposure grooming and mucociliary clearance from the upper respiratory tract.

Acrylic acid was rapidly absorbed following oral exposure and distributed throughout the body. The highest levels were found in the liver, fat, small intestine, brain and kidneys. Uptake from the stomach was biphasic with biological half-lives of 3.6 and 23 minutes, respectively (Kutzman et al., 1982).

Apparently, acrylic acid is metabolized rapidly *in vivo*, as indicated by the rapid appearance of $^{11}CO_2$ in the expired air (*vide infra*) (Kutzman et al., 1982); however, unlike its esters, acrylic acid does not form glutathione conjugates *in vitro* (Miller et al., 1981).

Kutzman et al. (1982) found that acrylic acid was rapidly excreted by rats following either oral or inhalation exposure, with 63-75% of the administered ^{11}C being excreted within 65 minutes. The urine accounted for 6% of the administered dose, the feces accounted for 9% and the remaining 48-60% was excreted as $^{11}CO_2$. The elimination of labeled CO_2 after either inhalation or oral exposure to acrylic acid was biphasic, with half-times for the α phase of 30.6 and 39.3 minutes, respectively. The termination of the study after 1 hour prevented the determination of the half-time for the φ phase.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of acrylic acid were not located in the available literature. Acrylic acid is not on the list of compounds to be tested by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

Lijinsky and Andrews (1980) tested acrylic acid in both plate incorporation and liquid preincubation assays using Salmonella typhimurium strains TA1535, 1537, 1538, 98 and 100. Tests were performed with or without rat liver and hamster liver S-9 preparations. The maximum concentrations that were tolerated without significant toxicity to the bacteria were 1000 µg in the plate incorporation assay and 250 µg in the liquid preincubation test. Acrylic acid was not mutagenic at any concentration tested under any of the assay conditions. Acrylic acid has, however, been demonstrated to inhibit the incorporation of thymidine into DNA and uracil into RNA in both Staphylococcus aureus and Escherichia coli (Glombitza and Heysen, 1971).

5.3. TERATOGENICITY

One reference was found in the literature relating to the teratogenicity of acrylic acid (Singh et al., 1972). Acrylic acid [1/10, 1/5 or 1/3 of the acute LD_{50} (22 mg/kg)] was administered to female Sprague-Dawley rats (5/group) by intraperitoneal injection on days 5, 10 and 15 of gestation. The rats were sacrificed on day 20 and then examined for embryonic-fetal toxicity, gross external malformations, skeletal malformations and fetal weight. No statistically significant effects were noted at the lowest dose level when comparisons were made with vehicle-treated controls, although 3 of 3 fetuses examined had elongated and fused sternebrae and frontal ribs.

A dose-related decrease in mean weight of fetuses was observed, which was reported to be statistically significant for all dosage groups when comparisons were made with untreated control animals. The incidence of total gross abnormalities was significantly elevated in the mid (3.7%) and high (9.1%) dose groups, and total skeletal abnormalities were significantly increased in the high dose group (16.7%) when compared to the incidence of gross and skeletal abnormalities [2 and 9.3% (average of three values), respectively] in the "volume control group" (a pooled control group consisting of animals treated intraperitoneally with distilled water, saline or corn oil). From the group given the highest dosage, 4 hemangiomas and 3 dead fetuses were found. Four of the 24 stained fetuses from this group had skeletal malformations consisting of elongated and fused sternebrae and/or frontal ribs. In the middle dosage group, 2 fetuses had hemangiomas of the neck, while 3 of 28 stained fetuses had elongated and fused sternebrae and frontal ribs.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of acrylic acid were not located in the available literature.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Gage (1970) exposed male and female Alderley Park specific-pathogen-free rats (4-8 rats/group) to an atmosphere containing 80 or 300 ppm acrylic acid, 6 hours/day, 5 days/week, for 4 weeks. The high dose level resulted in nose irritation, lethargy and retarded weight gain; however, all organs appeared normal at autopsy. No sign of toxicity was noted in the 80 ppm exposure group. All organs in both dose groups were classified as normal. In this study, a survey was made of a large number of industrial chemicals; detailed information on experimental procedures and results was not provided for the individual chemicals studied.

In an abstract of a Polish paper, Majka et al. (1974) noted irreversible changes in the skin and eyes of rats exposed to atmospheres containing 240 ppm acrylic acid, 4 hours/day for 5 weeks. Other toxic symptoms observed were reduced body weight gain and an increased number of blood reticulocytes.

5.6. OTHER RELEVANT INFORMATION

The acute toxicity of acrylic acid is summarized in Table 5-1. Based on the rat data, the oral and inhalation toxicities are equivalent; however, the LD_{50} for acrylic acid administered intraperitoneally is an order of magnitude lower. These differences are statistically significant ($p<0.05$, Students t-test) despite the large variance in the oral and inhalation data. The same difference between oral and intraperitoneal administration seems to occur in the mouse; however, the data are insufficient for a statistical test.

Gage (1970) exposed rats to atmospheres containing acrylic acid (see Section 5.5.). Short-term exposures, 5 hours at 6000 ppm or 4-6 hours at 1500 ppm, resulted in a number of pathological changes. In the high-dose group, all four of the rats displayed nose and eye irritation, respiratory difficulty and unresponsiveness. One of the four rats died. Histological examination revealed lung hemorrhage and kidney tubule and liver degeneration. The low-dose group (8 rats) exhibited nasal discharge, lethargy and weight loss. Histological examination revealed congestion in the kidneys.

Acrylic acid, as a liquid or concentrated solution or vapor, is a primary irritant, causing irritation and corrosion of the skin, mucous membranes, and eyes (Fassett, 1963; Nemec and Bauer, 1978). Ducombs et al. (1974) reported that exposure of workers to acrylic acid, due to defective polymerization of a printing ink, was related to the development of an irritant dermatitis of the hand.

TABLE 5-1

Acute Toxicity of Acrylic Acid

Species	Route	Result	Dose	Reference
rat	oral	LD_{50}	340 mg/kg	Carpenter et al., 1974
rat	oral	LD_{50}	193 mg/kg	Union Carbide Corporation, 1977
rat	oral	LD_{50}	2,520 mg/kg	Fassett, 1963
rat	oral	LD_{50}	2,100-3,200 mg/kg	Miller, 1964
rat	oral	LD_{50}	2,400 mg/kg	NIOSH, 1977
rat	intraperitoneal	LD_{50}	24 mg/kg	Majka et al., 1974
rat	intraperitoneal	LD_{50}	22 mg/kg	Singh et al., 1972
rat	inhalation	LC_{50}	18,000 mg/m³/5 hour	Gage, 1970
rat	inhalation	LC_{50}	3,600 mg/m³/4 hour	Majka et al., 1974
mouse	intraperitoneal	LD_{50}	128 mg/kg	NIOSH, 1977
rabbit	skin	LD_{50}	750 mg/kg	Union Carbide Corporation, 1977
rabbit	skin	LD_{50}	295 mg/kg	Carpenter et al., 1974
rabbit	skin	LD_{50}	950 mg/kg	Fassett, 1963

6. AQUATIC TOXICITY

6.1. ACUTE TOXICITY

The LC_{50} of acrylic acid in the golden orfe, Leuciscus idus, was 315.0 mg/ℓ (exposure period was not reported) (Juhnke and Luedemann, 1978). The 24-hour LC_{50} in the bluegill sunfish, Lepomis macrohirus, was 188 mg acrylic acid/ℓ (Dowden and Bennett, 1965).

In the water flea, Daphnia magna, the LC_{50} of acrylic acid for a 48-hour exposure period was 50 mg/ℓ (Dowden and Bennett, 1965). The mosquito larvae, Culex sp., were much more resistant with 24- and 48-hour LC_{50} values >1000 mg acrylic acid/ℓ (Dowden and Bennett, 1965).

The toxicity threshold values, determined by octanol cell counts (after 72 hours) or as a 3% decrease in turbidity (after 16 hours) for the protozoan, Entosiphon sulcatum, and the bacteria, Pseudomonas putida, were 20 and 41 mg/ℓ, respectively (Bringmann and Kuehn, 1980).

6.2. CHRONIC EFFECTS

Pertinent data regarding the effects of chronic exposure to acrylic acid on aquatic organisms were not located in the available literature.

6.3. PLANT EFFECTS

The toxicity threshold, measured turbidometrically as decreased cell growth after a 7-day exposure period of acrylic acid in the green algae, Scenedesmus quadricauda, was 18 mg/ℓ (Bringmann and Kuehn, 1980). Sullivan and Ikawa (1972) reported that acrylic acid at 1000 mg/ℓ completely inhibited algal growth (36 mm zone of inhibition growth in culture plate) and 100 mg/ℓ produced a zone of growth inhibition of 15 mm in UNH strain Chlorella pyrenoidosa. Strain 395 of C. pyrenoidosa was inhibited (35 mm zone of inhibition) at 1000 mg/ℓ, but strains 251 and 252 of C. pyrenoidosa and C. vulgaris species were not inhibited at this level of exposure (Sullivan and Ikawa, 1972).

6.4. RESIDUE

Pertinent data regarding acrylic acid residues in aquatic organisms were not located in the available literature.

6.5. OTHER RELEVANT INFORMATION

Additional data regarding the effects of acrylic acid on aquatic organisms or in aquatic environments were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

The use of acrylic acid is permitted by the U.S. Food and Drug Administration in the following forms: as polymers and copolymers; as a component of resinous and polymeric coatings; paper and paperboard components; adhesive; resin-bonded filters; and polyolefin films and rigid, semi-rigid and modified acrylic and vinyl plastics (5 wt % of the total polymer, maximum) intended for use in contact with food (21 CFR 176.180). The American Conference of Governmental Industrial Hygienists (ACGIH, 1980, 1982) has adopted a TLV of 10 ppm (~30 mg/m^3) in workroom air. This value is based largely on the study by Gage (1970) (see Section 5.5.) and is "recommended to prevent nasal and eye irritation and by analogy to glacial acetic acid."

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of acrylic acid were not located in the available literature.

8. RISK ASSESSMENT

No information regarding the carcinogenicity of acrylic acid was available. One study (Lijinsky and Andrews, 1980) indicated that acrylic acid was not mutagenic in S. typhimurium. Among the available toxicity data, the best study located was a 4-week subchronic inhalation study by Gage (1970). This study indicated a NOEL of 80 ppm (6 hours/day, 5 days/week); however, in terms of duration of the study, the data were judged insufficient for the derivation of an ADI.

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APPENDIX: LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

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